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PULMONARY ARTERIAL WALL SHEAR STRESS AND ITS IMPACT ON RIGHT VENTRICULAR FUNCTION IN PULMONARY ARTERIAL HYPERTENSION: PRELIMINARY ASSESSMENT BY COMPUTATIONAL FLUID DYNAMICS

Poster Contributions

Poster Sessions, Expo North

Sunday, March 10, 2013, 9:45 a.m.-10:30 a.m.

Session Title: Pulmonary Hypertension: Right Ventricle / Congenital Heart Disease

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Background: Pulmonary arterial hypertension (PAH) can lead to substantial morphometric remodeling of the pulmonary arteries. Previous studies have established coupling relationships between right ventricular (RV) function and pulmonary arterial hemodynamics. Our objective is to develop and validate a computational fluid dynamics (CFD) model to estimate spatially averaged wall shear stress (WSS) for patients with PAH, and explore correlations between WSS and known clinical metrics. We present a preliminary analysis of the first 6 patients to which the model was fully applied.

Methods: Computed tomography angiography (CTA) and right heart catheterization were used to retrospectively acquire images and hemodynamic data for 49 PAH patients. Each patient's pulmonary vascular tree was reconstructed from CT images up to 6 generations. The inlets and outlets were extended to 10 times their hydraulic diameters and a volumetric mesh created within element dimensions determined by a systematic mesh convergence study. Each patient model is simulated using a structured tree outflow boundary condition that uniquely corresponds to the pulmonary vascular resistance of that patient. Constant flow rate simulations were performed using the numerical solver Fluent (ANSYS Inc., Lebanon, NH) after adjusting for patient-specific scaling parameters. Finally, spatially averaged WSS on the surface of the pulmonary arterial endothelium were obtained during post-processing

Results: Computational simulations reveal an elevated spatial average WSS for patients with PAH: 15.4 ± 3.8 dyne/cm² (Mean \pm SD), with a strong correlation ($R^2 = 0.86$) between WSS and calculated PVR. Preliminary qualitative assessment does not show a definitive relationship between WSS and RV dysfunction.

Conclusions: Pulmonary arterial WSS obtained by CFD can be used as a physiologically relevant quantitative metric of PAH severity and progression. With established understanding of the impact of the underlying pathophysiology on WSS in the pulmonary vasculature, our CFD model shows promise as a reliable noninvasive tool to assess disease progression and response to PAH specific therapies.